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Daytime Sleepiness, Driving Performance, Reaction Time and Inhibitory Control during Sleep Restriction Therapy for Chronic Insomnia Disorder

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Short title: Consequences of sleep restriction therapy

Abstract

Background: Sleep restriction therapy (SRT) is a largely untested single treatment component of cognitive-behaviour therapy for insomnia. To date, the evidence for contraindications for SRT is limited to very few studies. The present study investigated the objective and subjective daytime consequences during the acute phase of SRT for adults diagnosed Chronic Insomnia Disorder.

Methods: Sixteen adults (age=36.3±13.4 yrs, 12 females, 4 males) underwent SRT for their insomnia over a 2-week period based on recommendations by Miller and colleagues (2014)[6]. Participants completed sleep diaries, self-reported daytime sleepiness (Epworth Sleepiness Scale [ESS]), as well as objective measures of reaction time/inhibition (Go/NoGo task) and driving performance (AusEd driving simulator) at pre-, mid- (i.e., after 1 week of SRT) and post-SRT (after 2 weeks of SRT).

Results: Sleep diary outcomes indicated participants complied with the restriction of time in bed, and that a similar amount of total sleep time (TST) was maintained from pre-to-post-treatment. There was no significant change in daytime sleepiness, and similarly no significant changes observed in objective performance on the Go/NoGo task and AusEd driving simulator.

Conclusions: These preliminary results suggest SRT during the acute phase does not appear to place insomnia patients at risk of significant impairments in sleepiness and reaction times. We note these findings can only be translated into clinical practice when sleep duration remains relatively unchanged. Future studies using objective measures of sleep and a control group are recommended.

Keywords: insomnia, sleep restriction therapy, sleepiness, sleep, reaction times, driving performance

Introduction

An estimated 20% of adults have trouble falling asleep, experience frequent awakenings during the night, and feel tired in the morning[1]. Despite this, only 6.9% of the population are diagnosed with Chronic Insomnia Disorder[1]. Insomnia is a sleep disorder characterised by the difficulty initiating and/or maintaining sleep, even when substantial sleep opportunity is available, and is often accompanied by daytime impairments (e.g., excessive daytime sleepiness, deficits in attention and concentration)[2,3].

At the present time, there are several well-established pharmacological, behavioural and psychological treatments available for insomnia. Cognitive-behavioural therapy for insomnia (CBT-I) uses of a combination of behavioural and psychological therapies to improve insomnia symptoms (e.g., cognitive therapy, stimulus control therapy) and for many years has been considered the gold standard treatment for insomnia[4,5]. One of the arguably most potent behavioural therapies within CBT-I is sleep restriction therapy[6].

In a recent meta-analysis, sleep restriction therapy (SRT) was established as an effective standalone treatment for insomnia that can achieve sleep improvements in fewer sessions than CBT-I[6]. SRT involves providing insomnia patients with a new sleep opportunity window that restricts their time in bed to the number of hours they currently spend asleep to avoid spending excessive amounts of time in bed (a key maintaining factor of insomnia)[6]. Restricting the opportunity for sleep to the average amount of time currently spent asleep (e.g., 12:00 AM to 6:00 AM if average sleep duration = 6 hrs of sleep per night) increases sleep homeostatic pressure across the course of the day/evening, resulting in more consolidated sleep, faster sleep onset latency, and increased sleep efficiency (i.e., percentage of time spent asleep whilst in bed)[6-8]. These sleep opportunity windows are often altered throughout treatment based on the previous week's sleep efficiency (SE) (i.e., ≥85% SE for the past week means an extra 30 min

of time in bed is afforded, ≤85% SE, 30 min less time in bed). However, an recent consensus states that sleep opportunity windows should not be reduced to <5 hrs a night to protect against excessive daytime sleepiness[6,9], since this is often reported as a prominent side effect during the acute phase of SRT[3,10,11].

During the acute stage of SRT (i.e., first couple of weeks), many recipients report experiencing negative daytime functioning side effects compared to pre-treatment[3]. One study that collected audio diary entries and conducted interviews with 18 insomnia participants undergoing SRT found that most reported feeling exhausted and irritable, having reduced motivation, and experienced difficulty with concentration and memory[3]. However, the study did not use any standardised measures of daytime sleepiness (e.g., Epworth Sleepiness Scale [ESS]). Of concerning to patients and clinicians is that more than one third of their sample self-reported compromised driving abilities while undergoing SRT (i.e., difficulty maintaining wakefulness, slowed reaction times, concentration impairment)[3]. Despite this, very few studies have measured driving performance in people undergoing SRT for insomnia.

A later study by Kyle et al. (2014)[11] administered the ESS to 16 insomnia patients receiving SRT and found *increased* daytime sleepiness in the initial 2 weeks of treatment. This daytime sleepiness was reflected in impaired objective performance on a reaction time task[11]. Although some studies have used tasks sensitive to attentional lapses such as the psychomotor vigilance Task (PVT)[11,12], there is a lack of literature focusing on inhibitory control and concurrent tasking (i.e., halting a pending thought or action to start another) for those undergoing SRT, which are arguably important cognitive mechanisms for safe driving[3].

Consequently, the current study sought to determine whether insomnia patients undergoing SRT experienced side effects that could impair their functioning (i.e., daytime sleepiness, reaction time, inhibitory control) and their driving performance. Despite SRT

potentially being used worldwide since 1987[9], to our knowledge there are no data on resultant driving performance. Thus, findings from this study will help to inform clinicians providing SRT of the potential risks resulting from the therapy, which ethically should be passed onto their patients. We anticipate that SRT will result in increased daytime sleepiness during the acute phase of treatment (first 2 weeks), as well as slower reaction times and reduced inhibitory control (Go/NoGo Task), and poorer driving ability (AusEd Driving Simulator).

Method

Participants

16 insomnia participants (mean age = 36.3±13.4 yrs, range 21-58, 12 females, 4 males) were recruited through advertisements on Facebook and flyers distributed throughout Flinders University and surrounding general practitioner/psychology clinics and community centres. Eligible participants met criteria for Chronic Insomnia Disorder based on the International Classification of Sleep Disorders, 3rd edition (ICSD-3) criteria[2]. Potential participants were excluded if they had a co-morbid sleep disorder associated with excessive daytime sleepiness (i.e., narcolepsy or sleep apnoea), had current suicidal ideation, or any other pre-existing conditions that could be exacerbated by sleep restriction (i.e., bipolar disorder, epilepsy or psychosis)[13-15], or did not possess a driver's licence. Ethics approval was granted from the Social and Behavioural Research Ethics Committee at Flinders University.

Materials

Diagnostic assessment measure

The diagnostic assessment was in the form of a semi-structured interview based on diagnostic criteria based on the ICSD-3[2], including sleep difficulties (Criterion A), associated daytime impairment (Criterion B), adequate sleep opportunity and sleep environment

(Criterion C), frequency (Criterion D) and chronicity (Criterion E) of nighttime/daytime symptoms, and differential diagnosis (Criterion F). Other information relevant for implementation of SRT was obtained, including napping habits, medications, and caffeine and alcohol intake (sleep hygiene).

Sleep Diary

Participants were asked to complete a pen-and-paper sleep diary daily, each week, over pre-treatment, and 2 weeks of SRT. The sleep diary assisted in confirming the insomnia diagnosis and differential diagnosis of other sleep disorders (e.g., circadian rhythm disorders)[16]. During treatment, sleep diaries were also used to check participants' compliance with time in bed instructions. The sleep diary provided subjective measures of bedtime, and final rise times, time of the sleep attempt, sleep onset latency, wake after sleep onset, time in bed, total sleep time, and sleep efficiency. Thus, the sleep diary used in the present study overlapped with the core consensus sleep diary[17-18]. Sleep diaries are a valid and reliable method for assessing changes in sleep and insomnia treatment outcomes[16,19].

Daytime Sleepiness

The Epworth Sleepiness Scale (ESS) is an 8-item questionnaire, used to assess daytime sleepiness in a variety of situations over the given day[20]. The ESS was used to screen for ineligible participants (ESS scores > 10), track daytime sleepiness, and used as a safety measure once treatment had commenced (ESS > 10 afforded an extra 30 min in bed to protect against excessive daytime sleepiness). Participants were asked to rate the likelihood of dozing (*0 would never doze, 3 high chance of dozing)* on each item, with higher scores indicative of greater levels of sleepiness. The ESS significantly correlates with the multiple sleep latency test, a validated objective measure of sleepiness[20].

Objective daytime functioning measures

Reaction Time / Inhibitory Control

The Go/NoGo task is a computerised test used as a measure of speed accuracy and inhibition[21]. Participants were required to respond to "Go" stimuli (i.e., the letter 'M') appearing on a computer screen as quickly as possible by tapping the spacebar, yet avoid pressing the spacebar when a "No" stimulus was presented (i.e., the letter 'W'). Each letter was displayed for 0.216 sec and the interval between each trial varied between 1300-1700ms, with the task lasting 4 min. If participants failed to identify a "Go" stimulus after 500ms, a tone sounded, signalling they had missed the stimulus. Reaction times measured their correct decision speed, with additional measures of omission accuracy (responses to Go, 'M') and commission accuracy (responses to NoGo, 'W')[22-23].

Driving Performance

The AusEd driving simulator is a computer-based task designed to measure driving performance[24]. A standard PC computer was fitted with a steering wheel and brake/acceleration apparatus. Steering deviation (in centimetres), speed deviation (in km/h), reaction time (ms) and crash frequency (number of crashes) were recorded over a 15-min driving simulation. Participants were required to complete a 5-min practice drive to familiarise themselves with the simulator before each 15-min drive. Participants were required to drive between 60-80km/h and brake completely (i.e., bring car to complete stop) when a truck appeared onscreen, and resume driving once the truck disappeared. The task simulates driving on a country road at night with features such as engine noise to simulate a real-world driving experience. The AusEd driving simulator is sensitive to performance deficits as a result of various factors, including sleep deprivation[24].

Procedures

Treatment was manualised and based on Spielman et al.'s (1987)[9] original depiction of SRT and a consensus by Miller et al. (2014)[6]. SRT was conducted over a 2-week period at the Flinders Insomnia Clinic by clinical, registered and provisional psychologists (mean experience= 4 yrs 4 mo). The first session involved an assessment to establish study eligibility, followed by psychoeducation about SRT (i.e., the premise of sleep homeostatic pressure), and the implementation of a new tailored sleep schedule. Sleep schedules were designed in accordance with participants' pre-treatment sleep diaries (i.e., restricting time in bed to equate to their average pre-treatment total sleep time), yet prescribed no less than 5 hrs per night (Miller et al., 2014)[6]. Immediately after this session (ie, prior to restriction of time in bed), insomnia participants' completed the ESS and were invited to complete computerised testing in order to track changes in objective daytime functioning. The computerised tasks were conducted in a dimly-lit (<4 lux) sound-attenuated room in the Flinders University Sleep Laboratory, and included the Go/NoGo Task and the AusEd driving simulator.

The following weekly sessions were used to review treatment progress and titrate time in bed based on sleep efficiency. Specifically, if sleep efficiency was >85%, or if ESS scores were >10, then this afforded the opportunity to extend the amount of time in bed by 30 min (Miller et al., 2014)[6]. Following each session, participants once again completed the ESS, Go/NoGo Task, and AusEd driving simulator. After the 2 weeks of SRT, insomnia participants were offered other treatment components of CBT-I (e.g., stimulus control therapy, sleep hygiene, cognitive therapy).

Results

Manipulation Check and Sleep Outcomes

Linear mixed-model (LMM) regression analyses were performed on time in bed (TIB) to establish whether insomnia participants complied with SRT instructions, as well as total sleep time (TST) to observe whether sleep had reduced following SRT. LMMs were employed as opposed to ANOVAs as the former analytical technique has the ability to model change in missing data points (i.e., compensate for missing data)[25]. An AR(1) model was used in LMM's as the study employed a repeated measures time-based model and to also compensate for variability in smaller samples[26].

Insomnia participants complied with a reduction in TIB, evidenced by a significant decrease in over time $F(1,2)=8.52$, $p=.002$. As seen in Table 1, TIB decreased significantly by a mean of 1 hr and 10 min from pre- to mid-treatment, and 1 hr and 23 min from pre- to post-treatment. However, despite a mean TST decrease of 32 min from pre- to post-treatment, TST did not significantly change during SRT, $F(1,2)=2.56$, $p=.10$. Sleep onset latency significantly reduced from pre-treatment (51 min) to post-treatment (23 min), $F(1,2)=7.56$, $p=.004$, as did wake after sleep onset (WASO; pre-treatment=75 min to post- treatment=38 min), $F(1,2)=5.67$, $p=012$. However, there was no significant change in sleep efficiency, $F(1,2)=1.33$, $p=.29$.

< Insert Table 1 about here >

Daytime functioning outcomes

No significant changes in ESS scores occurred during the study, $F(1,2) = 0.84$, $p=.45$, indicating chances of dozing remained somewhat constant. Similarly, there were no significant

changes in reaction times, $F(1,2)=2.75$,$p=.09$, commission accuracy $F(1,2)=0.25$, $p=.78$, or omission accuracy $F(1,2)=0.33$, $p=.72$ on the GONOGO reaction time/inhibitory task. Likewise, there were no significant changes to various domains of driving ability, including reaction times, $F(1,2)=0.60$, $p=.56$, speed deviation, $F(1,2)=0.58$, $p=.57$, steering deviation, $F(1,2)=1.82$, $p=.19$, or crash frequency, $F(1,2)=0.00$, $p=.99$, on the AusEd driving simulator.

< Insert Table 2 about here >

Discussion

The aim of the current study was to investigate subjective and objective changes in daytime functioning and performance during the acute phase (first 2 weeks) of sleep restriction therapy (SRT). Despite insomnia participants' compliance with SRT, no significant changes were observed in reaction times, inhibition, driving performance variables or daytime sleepiness during the acute phase of treatment. These findings suggest SRT for the treatment of insomnia does not appear to negatively impact driving performance or other aspects of daytime functioning that would place patients at risk regarding their safety.

Insomnia participants adhered to the SRT protocol (as per Miller et al., 2014)[6] as evidenced by a significant 81-min reduction in TIB (from 8 hrs 41 min to 7 hrs 20 min). From week 1 to week 2 of SRT there was no significant change in TIB indicating similar sleep schedules were maintained across the treatment period. Despite this significant reduction in TIB, there was no significant change in TST across treatment. We do note though a mean decrease of 32 min in TST, which may be a meaningful reduction of sleep duration from pre-treatment. Therefore, we now discuss the outcomes from the perspective of this *mild* sleep restriction.

We anticipated that self-reported daytime sleepiness (ESS scores) would increase from pre-treatment to post-treatment (i.e., the acute 2 weeks of SRT). However, this was not observed. This prediction was based on the premise that insomnia patients may have experienced reductions in TST, as well as results from previous studies[11]. The lack of change in our participants' self-reported daytime sleepiness, even in response to a mean 32-min reduction in TST, aligns with previous studies of stable daytime sleepiness after 1 week of ~30-min less TST in good sleepers[27]. In contrast, TST reductions >1hr in good sleepers[28] and insomnia patients[11] over an approximately 2-week period produce increases in daytime sleepiness. Taken together, the findings suggest that a reduction of ~30-min of TST during the acute phase of SRT (ie, first 2 weeks) is not potent enough to result in changes in patients' perceived daytime sleepiness. If anything, we note no change after 1 week of SRT (*d*=0.02), yet a small *decrease* (*d*=0.44) from mid-treatment (i.e., after 1 week of SRT) to post-treatment (i.e., after 2 weeks of SRT), suggesting a reduced risk for insomnia patients undergoing SRT. However, an important contribution from the present study is systemically examining multiple outcome measures, so as not to rely solely on patients' ESS scores.

The Go/NoGo task was an objective test of participants' reaction times and inhibition[21]. Although previous studies found increased reaction times (using the PVT) following sleep restriction in good sleepers[12,29] and from SRT for insomnia[11], the present study did not find such deficits. Even in the context of a 32-min TST reduction, effect sizes during SRT were virtually nil (*ds*=0.00-0.03). Similarly, both omission and commission accuracy did not significantly change, with negligible effects for omission accuracy (i.e., accidently responding to 'NoGo' stimulus; *ds*=0.06-0.10). Although small effects were found for omission accuracy (i.e., correctly responding to 'Go' stimulus; *ds=0.23-0.27*), the actual percentage change was within 1% which is not meaningful).

Driving performance was the second objective task used during the acute phase of SRT, as insomnia patients undergoing SRT self-report driving difficulties[3] yet to our knowledge this has not been objectively verified. Contrary to expectations, reaction time, speed deviation, steering deviation and crash frequency did not deteriorate over the treatment period. Although small effects occurred during SRT (i.e., Δreaction time=0.06ms; Δsteering deviation=4cm; Δspeed deviation<0.2km/hr), these are not meaningful. Previous studies using the AusEd driving simulator have reported deficits in driving parameters, however these were observed following a significant reduction in total sleep time and/or consumption of alcohol in good sleepers (e.g., TIB=5 hrs)[30,31]. Given insomnia patients' self-reports of impaired driving whilst undergoing SRT, more data are required to more confidently claim that it is safe for such patients to drive during the acute phases of treatment.

Implications

The present study provides preliminary evidence to suggest SRT may be a safe clinical intervention for people with insomnia. The chances of dozing, reaction times, inhibitory control and driving performance remained relatively stable after 2 weeks of SRT. This appears to occur despite a reduction of ~30-min (or less) sleep duration. Insomnia patients' self-reports of driving performance may not mirror objective assessments of driving performance, which supports theoretical models of distorted daytime performance deficits[32]. However at this stage, we reserve the use of these preliminary findings to psychoeducation (over behavioural experiments) during treatment. Although we did not objectively measure TST during SRT like previous studies[11], the use of polysomnography or wrist actigraphy is arguably not the norm in clinical practice, whereas sleep diaries are more accessible. This suggests the findings from the present study are applicable broadly to clinicians performing SRT. Likewise, while our sample may be considered small (N=16), it is comparable to the two other studies of SRT as a stand-

alone treatment for insomnia (N=16-18)[3,11], and it included a range of mental health and medical co-morbidities that increases the chances of these implications being applicable in clinical practice[33-35].

Limitations and Future Directions

Research to answer the question "*Is sleep restriction therapy safe?*" would be enhanced by further replication, using sample sizes larger than that in the present study (N=16) and others (N=16-18)[3,11], and including a control group (e.g., waitlist control). We do not necessarily suggest larger samples to be tested in order to detect statistical significance (as we believe the meaningfulness of changes is important), but more so that findings can be generalised better to insomnia patients undergoing SRT. It is not exactly known how much sleep patients obtained during the present study, as no objective measures were used[11,36-37]. Therefore, future studies are recommended to use both subjective (sleep diary) and objective (wrist actigraphy) measures of sleep quantity during SRT. Simultaneous measurement would also address the issue that a significant proportion of people with insomnia experience sleep misperception (i.e., under-estimate TST)[38], which could lead to more accurate implementation of SRT. We also note that the present study used a 15-min driving simulator, so future studies should consider extending the duration (e.g., 60 min)[24]. This will give an indication as to whether or not sustained attention whilst driving deteriorates, or whether it remains the same over a longer period of time.

Conclusions

Despite sleep restriction therapy (SRT) being available for clinicians and insomnia patients since 1987[9], it is surprising that after 30 years that there are very few data directly testing associated impairments, and thus safety. The present study's findings suggest when

adhering to recent guidelines (Miller et al., 2014)[6], SRT does not appear to negatively impact daytime functioning and driving performance. This lack of effect assumes a nil-to-mild reduction of total sleep time during treatment (i.e., less than 30 min). However, future investigations of SRT as a stand-alone treatment are needed, with preferably a larger sample size and control group in order to make clearer judgements for people with Chronic Insomnia Disorder.

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Table 1. Changes in sleep outcomes from pre- to mid- to post-treatment.

	Time					
	Pre		Mid		Post	
	M	CI	M	CI	M	CI
Sleep outcomes						
TIB (hrs)	8.68	7.97-9.39	7.43	6.72-8.14	7.33	6.57-8.1
TST (hrs)	6.30	5.55-7.05	5.83	5.08-6.58	5.77	5.00-6.55
WASO (mins)	75.58	51.69-99.48	38.52	14.62-62.42	38.4	11.08-65.72
SOL (mins)	51.09	37.36-64.82	26.1	12.37-39.83	23.86	37.36-64.82
SE (%)	73.19	65.55-80.84	79.01	70.68-87.35	79.21	71.57-86.86

Note: *Pre= pre-treatment; Mid = after 1 week of SRT; Post = after 2 weeks of SRT. TIB = time in bed; TST = total sleep time; WASO = wake after sleep onset; SOL = sleep onset latency; SE = sleep efficiency; M = estimated marginal mean; CI = 95% confidence interval.*

Table 2. Changes in daytime sleepiness and objective performance from pre- to mid- to post-treatment.

	Pre		Pre-mid	Mid		Pre-post	Post	
	M	CI	*d*	M	CI	*d*	M	CI
ESS								
Sleepiness	7.00	4.28-9.72	*0.02*	7.12	3.34-10.95	*0.41*	4.46	0.54-8.37
GO/NOGO								
Reaction times (ms)	0.34	0.31-0.36	*0.00*	0.33	0.31-0.35	*0.03*	0.35	0.35-0.37
Commission accuracy (%)	75.51	64.79-86.23	*0.10*	77.65	66.2-89.10	*0.06*	74.46	63.88-85.05
Omission accuracy (%)	99.48	99.08-99.88	*0.23*	99.66	99.23-100.09	*0.27*	99.70	99.25-100.15
AusEd								
Reaction time (ms)	0.97	0.85-1.08	*0.09*	0.95	0.83-1.06	*0.36*	0.89	0.76-1.02
Steering deviation (cm)	30.87	23.10-38.63	*0.30*	35.19	27.43-42.95	*0.29*	35.12	26.97-43.28
Speed deviation (km/h)	0.85	0.42-1.28	*0.21*	0.68	0.25-1.11	*0.11*	0.76	0.28-1.23
Crash frequency	0.00	0.00	-	0.00	0.00	-	0.00	0.00

Note: *Pre= pre-treatment; Mid = after 1 week of SRT; Post = after 2 weeks of SRT. ESS= Epworth Sleepiness Scale. Cohen's d effect size magnitude: .20 = small, .50 = moderate, .80 = large. CI = 95% confidence interval.*

Highlights

- Sleep restriction therapy (SRT) is an effective treatment component of cognitive-behaviour therapy for insomnia
- Restriction of sleep can induce excessive daytime sleepiness and reaction times.
- Applying SRT as per recent guidelines led to a mean decrease in sleep duration of 32 minutes.
- No significant changes in sleepiness, reaction times or driving performance were found whilst insomnia patients underwent 2 weeks of SRT.